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AGRICULTURE

DISEASES OF VEGETABLES IN ALBERTA

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All outdated copies should be discarded.

FOREWORD

This handbook is designed to assist Alberta market garden and commercial vegetable growers in the diagnosis and treatment of diseases in vegetable crops. This publication is not intended to be a comprehensive treatment of all of the known disease problems on all vegetables; rather, it includes information on the major diseases likely to be encountered. In difficult or questionable situations, the opinion of a professional plant pathologist should be sought. Control recommendations, particularly those involving chemical pesticides, are subject to periodic change. Reference to pesticides in this publication is for the reader's convenience. Persons using such products assume responsibility for their use in accordance with current label directions of the manufacturer.

Additional copies of this publication can be obtained from: Library, Alberta Horticultural Research Center, Brooks, Alberta, TOJ OJO.

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ASPARAGUS

CROWN ROT AND SEEDLING BLIGHT

PATHOGEN: *Fusarium* spp.

SYMPTOMS: This disease is characterized by poor seedling establishment. Seedlings exhibit collapse of the primary roots and show foliar symptoms such as stunting, yellowing and wilting. Under some conditions, infected young plants may recover and grow almost normally. Infected established plants will produce weak spindly spears in the spring. Shoots become wilted, dwarfed and sometimes show a dingy brown color. As the season progresses, shoots from severely infected crowns may exhibit a brilliant yellow coloration and may posses a limited amount of vascular discoloration. Feeder roots are frequently rotted and discolored with the discoloration extending into the storage roots. Red or brown elongated spots may appear on asparagus spears, shoots or needles. Successive years of infestation reduces the vitality of the root system so that only cull shoots are produced or the whole plant dies.

DISEASE CYCLE: Plants produced under ideal conditions for good growth are rarely, if ever, infected. Excessive extension of harvest, flooding, drought, soluble salts, insufficient fertilizer or minor elements, injury from cultivation or herbicides, beetle injury or freezing produce conditions where the disease can become destructive. Fungus levels in infested soil will increase if asparagus is continuously grown without crop rotation. *Fusarium* spp. produce great masses of mycelium with sickle-shaped spores and may live as a saprophyte on humus in the soil for several years. Poor stand establishment will occur if new crops are planted in previously infested soil. The temperature requirements of the various *Fusarium* species extends over the entire range in which asparagus can grow. Disease appears worse during hot, wet weather and when the soil pH is acid.

CONTROL:

1. Destroy diseased beds and start a new planting in soil where asparagus has not been grown for at least 4-5 years. Locate new beds as far away as possible from mature beds.
2. Plant disease-free crowns, if possible.
3. Rotate affected fields with cereals or grasses.
4. If disease-free soil is not available for growing seedlings, disinfest the seedbed with methyl bromide, Vorlex or other suitable fumigants prior to planting.
5. Before transplanting, dip crowns in 1000 ppm benomyl solution for 15-20 minutes.
6. Viking 2K is resistant to fusarium crown rot.

RUST

PATHOGEN: *Puccinia asparagi*. This rust attacks not only the common edible asparagus, but also *A. maritimus*, *A. scaber* and *A. acutifolius* and slightly *A. verticillatus*. A number of other asparagus species are immune. The same fungus also infects onion, Welsh onion, shallot and chives. Leeks, garlic and wild leeks seem immune.

SYMPTOMS: Red or brown elongated spots may appear on asparagus spears, shoots or needles. Successive years of infestation reduces the vitality of the root system so that only cull shoots are produced or the whole plant dies.

DISEASE CYCLE: The life history of *P. asparagi* is composed of the four common rust stages, all of which are found on the one host. The first two spore stages take place in the oval light green patches on the first stalks that come through the ground. These lesions occur most abundantly in April and May, although some have been found as late as July. The relatively large spores produced (aeciospores) are blown by wind or washed by rains. The spore germinates in the presence of moisture and penetrates the asparagus plant. Once established, a fruiting layer of the fungus is formed just below the epidermis, which splits and releases brick red spores (urediospores) for dispersal. The spores are produced in such great numbers that when disseminated by the wind they cover the plants and ground with a reddish dust. They germinate at once in the presence of moisture, and within 12 days after inoculation may cause infection and yield a new generation of spores. The fungus requires droplets of water for germination. Heavy rains may wash off the infective basidiospores.

Later in the season, usually when cooler weather has set in or following a prolonged drought, another form of spore is produced. These large heavy-walled black spores cling to the old stems or stubble until spring. On germination, these spores give rise to the thin basidiospores which infect the newly emerged asparagus spears. The cycle is then begun for another year. If the young shoots are cut for the market, the pathogen does not have an opportunity to develop and the cycle is broken.

Plants growing in relatively dry soil are injured much more by the disease than those planted in wet soil.

CONTROL:

1. Avoid planting in areas having poor air circulation.
2. Cut or destroy all volunteer asparagus plants around fence lines, etc.

3. Irrigate early in the day so that plants do not remain wet overnight.

4. Grow cultivars with some resistance to rust (see page 58).

5. After harvesting has been completed, but before the disease appears, spray fern growth every 10-14 days with zineb (Dithane Z-78 or Zineb) or metiram (Polyram) to prevent infection.

BEANS

BACTERIAL BLIGHTS

PATHOGENS: Various bacteria: Common Blight - *Xanthomonas phaseoli*, Fuscous Blight - *Xanthomonas phaseoli* var. *fuscans* Halo Blight - *Pseudomonas phaseolicola*.

SYMPTOMS: Each blight, although caused by a different organism, produces similar symptoms on leaves, stems and pods. The symptoms are striking on bean leaves and start as small, water-soaked spots which enlarge, turn brown and finally kill the leaf. In halo blight, under cool conditions, a greenish-yellow zone forms around each lesion. In time, the disease may progress and cause every leaf on the plant to die and fall. Stem lesions appear as reddish logitudinal streaks weakening the stem and causing the plant to fall over. In humid weather, bacterial exudate may appear on lesions on any plant parts. On pods blight damage may be severe. Small, water-soaked spots enlarge and become surrounded by a reddish-brown border. The bacteria may infect the upper suture of the pods and invade the seeds, particularly if pods become infected while still young.

DISEASE CYCLE: Plants grown from infected seed develop lesions on cotyledons and stems, and the bacteria are splashed by rain from there to leaves of healthy plants. Bacterial blights are explosive diseases which can increase rapidly under favorable conditions. A few infected plants in a field, as a result of infected seed, may be the source of a general outbreak. Hail can predispose plants to infection. The bacterial blight organisms can also survive on infected crop debris in the soil for one or two years.

CONTROL:

1. Plow under crop refuse promptly after harvest.
2. Follow a 3-year rotation out of beans.
3. Avoid cultivation or working in bean fields during wet weather or in the morning whn dew is still on the plants.
4. Avoid sprinkler irrigation.
5. Plant seed which is certified free of bacterial blight.
6. If practical, apply protective sprays of tribasic copper sulphate (Tri-Cop) or cupric hydroxide (Kocide 101 or Coppercide). Preharvest interval = 1 day for both chemicals.

GRAY MOLD

PATHOGEN: *Botrytis cinerea*

SYMPTOMS: The most conspicuous symptom of this disease is felty, gray growth of the causal fungus on plant tissue. Resistant structures (sclerotia) may also form on infected plant parts. Sclerotia are usually small-sized, flattened and black in color. They are not always conspicuous, but may be embedded in decayed tissue or coated with soil and other debris. Gray mold affects bean plants in a number of ways. It may cause blossom blight, pod and stem rot or a moldy deterioration of the seed. The first symptom is usually a water-soaked spot which later becomes soft and watery. When extensive infection of the stems and branches occurs, plants may wilt and die prematurely. If humidity remains high, a grayish-brown web of fungus mycelium and spores develop over the infected tissues.

DISEASE CYCLE: Sclerotia formed on infested plant debris from previous crops usually constitute the initial source of infection. Under favorable conditions, sclerotia germinate and colonize plant debris on or below the soil surface via mycelial growth. Spores produced on above-ground debris can be spread by wind, spashing water or machinery to surrounding bean plants. A film of water is necessary for spores to germinate and infect plant tissues. Although germinating spores are capable of penetrating bean plants directly, they usually infect fallen petals or plant debris in contact with the stem, branches or pods and then grow into the healthy tissue. The fungus can produce a new crop of spores in a few days under favorable conditions. Sclerotia usually form on tissues where infection has been established for some time and enable the fungus to survive long, dry or extremely cold periods.

CONTROL:

1. Turn under infested crop debris to enhance decomposition.
2. Avoid growing beans after beans in a crop rotation.
3. Avoid irrigating after the rows close over.
4. Plant rows into the direction of prevailing winds to enhance air circulation between rows.
5. Plant varieties with a compact growth habit, which allows better air circulation through the canopy and thereby provides less favorable conditions for gray mold development.

6. Apply protective sprays of benomyl (Benlate), iprodione (Rovral) or dichloran (Botran) at 50% bloom. Thorough coverage of blossoms is essential. Preharvest interval = 2 days (dichloran), 14 days (benomyl). Rovral should not be applied beyond the flowering stage of beans.

ROOT ROT AND SEEDLING BLIGHT

PATHOGENS: Various fungi, principally *Pythium* spp., *Rhizoctonia solani* and *Fusarium solani* f. sp. *phaseoli*.

SYMPTOMS: Failure of plants to emerge from the soil indicates seed decay or seedling damping-off. Young plants may wilt and die after emergence or remain stunted and yellow (seedling blight phase). Reddish to dark brown rotted areas are seen on the taproot at or below the soil line. On larger plants, the disease often appears as a rusty brown discoloration of the taproot (root rot phase). As it progresses, the discolored area spreads until the entire taproot and lower stem are reddish brown and decayed. Secondary (branch) roots are usually decayed as well. Above-ground symptoms may not occur unless the degree of root rot is moderate to severe in which case there is likely to be stunting, yellowing of the foliage, wilting, some premature defoliation and a failure to produce normal, full pods.

DISEASE CYCLE: The causal fungi are all soil-borne. Plants may be attacked at any stage from seed through maturity. Infection usually occurs early in the season and symptoms become progressively more pronounced. Fields that have grown beans for several years are likely to be the most severely affected by root rot and seedling blight diseases. *Pythium*, *Rhizoctonia* and *Fusarium* are all capable of producing resistant structures which can survive in the soil for a year or more. These structures can be spread by running water, wind erosion and in dust and plant debris mixed with threshed seed. *Pythium* tends to be favored by cool, wet soil and is principally a seed rot and damping-off pathogen. *Rhizoctonia* and *Fusarium* prefer warmer soil conditions. *Fusarium* is especially severe in dry, compacted soils which are light in texture.

CONTROL:

1. Follow a crop rotation with cereals, alfalfa or corn that includes beans only once every five years.
2. Prepare the soil in a way that allows good moisture and root penetration.
3. Avoid practices which increase soil compaction and cause drought or poor drainage.
4. Light, frequent irrigations encourage new roots to form near the soil surface; however, this practice should be avoided if sclerotinia stem rot or bacterial blights are problems. Seed at rates that are high enough to ensure a good stand.
5. Treat seed prior to planting with captan or thiram fungicide to prevent damping-off.

SCLEROTINA WHITE MOLD

PATHOGEN: *Sclerotinia sclerotiorum*

SYMPTOMS: First indications of this disease are small, water-soaked areas on the stem, leaves or pod near or in contact with the soil. The growth and spread of infection is very rapid, under favorable conditions, and whole plants may be killed in the span of just a few days after symptoms are first observed. Plant tissues invaded by the fungus become soft and slimy. If the humidity and temperature remain high, a cottony white fungus growth soon becomes apparent on infected plant parts. This symptom has led to the common names "white mold" and "cottony soft rot" for this disease. Plants with stem infections generally wilt and die, while others with pod or branch lesions may survive. The slimy, water-soaked areas eventually dry up and turn brown. Grayish black resting bodies (sclerotia) of varying sizes are formed on the outside or within these dead plant parts.

DISEASE CYCLE: Sclerotia of the causal fungus are the principal means of carryover of the fungus in the soil. Seeds colonized with fungus mycelium may also serve to spread the disease. A great number of other broadleaved crops and weeds serve as natural hosts for *Sclerotinia*. During the season, under favorable conditions, sclerotia may germinate to produce mushroom-like structures which, in turn, produce spores. The spores are readily air borne and may cause direct plant infections. Generally, this mode of infection is less common than direct attack from mycelium in the soil. *Sclerotinia* can persist in the soil for many years.

CONTROL:

1. Avoid dense foliage by restricting nitrogen fertility and moisture levels.

2. Rotate with non-susceptible crops such as beets, onions, spinach, corn, cereals and grasses.

3. Apply protective sprays of benomyl (Benlate), dichloran (Botran) or iprodine (Rovral) at 50% bloom. Thorough coverage of blossoms is essential. Preharvest interval - 2 days (dichloran), 14 days (benomyl). Rovral should not be applied beyond the flowering stage of beans.

4. Plant rows into the direction of prevailing winds to enhance air circulation between rows.

5. Plant varieties with a compact growth habit, which allow better air circulation through the canopy and therby provide less favorable conditions for gray mold development.

COLE CROPS (BROCCOLI, BRUSSELS SPROUTS, CABBAGE & CAULIFLOWER

BLACKLEG

PATHOGEN: *Phoma (Plenodomus) lingam*

SYMPTOMS: Symptoms usually appear 2-3 weeks before transplanting. Well-defined ashen-gray spots occur on the leaves and similar spots surrounded by a purplish border occur on stems. If large areas of stem tissue are killed, many of the affected plants will die.

DISEASE CYCLE: The fungus is carried on the seed and persists on plant refuse for one or two years. The disease usually starts in the field from infested soil or seed. Sprinkler irrigation and rain may spread the organism.

CONTROL:

1. Practice a 4-year crop rotation with non-cruciferous crops.
2. Soil used for transplants and seeding should be sterilized with heat, chloropicrin or Vapam.
3. Use disease-free or hot water treated seed (See Black Rot page 10).
4. Maintain good soil drainage.
5. Keep fields free of cruciferous weeds such as stinkweed, shepherd's-purse and wild mustard.
6. Clean up crop debris or plow it down to aid decomposition.
7. Avoid cultivating fields when the plants are wet.

BLACK ROT

PATHOGEN: *Xanthomonas campestris*

SYMPTOMS: V-shaped dead areas appear at the leaf margins where infection usually occurs. Veins running from the point of infection are black and often extend to the stem of the plant. As the bacterium moves down the stem, younger leaves become infected.

DISEASE CYCLE: The bacterium is seed- and soil-borne. The organism can be transported by insects, splashing rain, sprinkler irrigration and farm equipment. Continuous cropping may build up the level of bacteria in the field and increase the chance of infection.

CONTROL:

1. Use hot-water-treated seed. Place seed loosely in a cheesecloth sack and submerse in water at 50°C for 25 min for cabbage and brussels sprouts, or 15 min for cauliflower or broccoli. Then immerse the seed in cold water, drain and spread in a thin layer to dry.
2. Practice a two-year crop rotation with non-cruciferous crops in fields.
3. Use a three-year rotation in the transplant bed. This is important since most spread of the disease occurs in the seed bed.
4. Destroy all diseased refuse after harvest, if practical.
5. Do not work in fields when the foliage is wet.
6. Control cabbage root maggots, cutworms and other insects which may spread the disease.
7. Plant resistant varieties (see pages 58-59).

CLUBROOT

PATHOGEN: *Plasmodiophora brassicae*

SYMPTOMS: The first symptoms of the disease in the above-ground parts of the plants are mild and difficult to distinguish. The leaves are pale green to yellowish and may show flagging and wilting in the middle of hot sunny days, sometimes recovering at night. Affected plants show almost normal vigor at first, but then gradual or sudden, pronounced stunting sets in, which may be followed by death of the plant. Young plants may be killed by the disease within a short time after infection, while older plants may remain alive but fail to produce marketable heads.

The most obvious symptom appears on the roots and sometimes the underground part of the stem. The symptom consists of knobby or club-shaped swellings on the roots and rootlets. These malformations may be individual or coalesce and cover the entire root system. Breakdown may occur upon infection by secondary microorganisms.

DISEASE CYCLE: The fungus overwinters in the soil and can remain there indefinitely. Once a field becomes infested, it becomes unfit for cultivation of cruciferous plants, unless costly methods and materials are used to sterilize the soil.

CONTROL:

1. Avoid growing cruciferous plants in clubroot-infested fields or, where clubroot is known to occur, rotate with other than cole crops at least 3 out of every 4 years.

2. Addition of hydrated lime (approximately 4.5 tonnes/ha) at least six weeks before planting can be used to adjust soils of acid pH to a slightly above neutral (pH 7.2) range. Spores of the clubroot organism germinate poorly in alkaline media.

3. Although very costly, seedbeds can be fumigated with chloropicrin, methyl, bromide or Vapam approximately two weeks before planting. Clean, clubroot-free seedlings, upon transplanting, can be watered with a solution containing 28 g of Terrachlor 75% WP [PCNB (pentachloro-nitrobenzene)] per 4.5 L of water.

4. Resistant varieties of cabbage, turnip and rutabaga are available for some races of the clubroot organism.

SCLEROTINIA ROT

See CARROTS - Cottony Soft Rot

SOFT ROT

See CARROTS - Bacterial Soft Rot

WIRESTEM AND DAMPING-OFF

PATHOGENS: *Rhizoctonia solani* and *Pythium* spp.

SYMPTOMS: Wirestem and damping-off are common fungal diseases in seedbeds. Seedling stems above and below the soil line turn brown or black in color. Affected tissues shrink and the stem becomes thin (wire-like), tough and woody. Affected seedlings transplanted to the field grow slowly and may eventually die. With damping-off, affected seedlings die before or shortly after emergence.

DISEASE CYCLE: Both fungi are soil borne and can persist in fields and seedbeds for many years.

CONTROL:

1. Grow seedlings for transplanting in steamed or fumigated soil.
2. Seedlings affected by wirestem can be detected when they are pulled out of the seedbed for transplanting and they should be discarded.
3. Delay planting as long as possible to avoid cold moist soils that favor disease development.
4. Avoid excessive irrigation.
5. Space seedlings in seedbeds to allow good air ciculation around plants.

CARROTS

ALTERNARIA AND CERCOSPORA LEAF BLIGHTS

PATHOGENS: *Alternaria dauci* and *Cercospora carotae*

SYMPTOMS: Alternaria blight: Early infection of the seedlings causes damping-off. On the foliage of mature plants, the lesions are dark brown to black and edged with yellow, first appearing at the margin of the leaflets. The leaflets may shrivel and die, which makes the plants appear blighted. Because older leaves are more frequently attacked than younger ones, the disease appears somewhat later than cercospora blight. The fungus may also attack the roots after mechanical injury. The root lesions are irregular, dark brown to black and produce a shallow, firm decay.

Cercospora blight: This disease affects the leaves, leaf stalks, stems and floral parts, but not the root. Lesions first appear along the margins of the leaves and cause the edges to curl. Spots inside the leaf edges are small, roughly circular, tan or gray to brown or almost black, with a dead center surrounded by a yellow area with no clear border. As the spots increase in number and size, the entire leaflet withers and dies. At this stage, the symptoms may be confused with those of alternaria blight. On the leafstalks and stems, the lesions are elliptical, tan to brown, with a paler center. In humid weather the spots are darker. Sometimes the lesions may merge and girdle the stem causing the stems to break and the leaves to die. As infected carrot tops tend to break easily, mechanical harvesting becomes a difficult operation. When the floral parts are attacked early, they shrivel before the seed is borne, but later attacks may permit the fungus to penetrate the seed without showing symptoms.

DISEASE CYCLE: Alternaria blight: The pathogen subsists on or in the seed and on diseased crop debris in the soil. The spores and mycelium produced are spread by wind, running water, splashing rain and moving objects. Moisture is essential for infection to occur. Cool weather is most favorable for the development of alternaria blight, so it is more likely to occur later in the season. This contrasts with cercospora blight, which usually appears earlier.

Cercospora blight: Between crops, the organism lives on or in the seed and on infected crop refuse. The spores produced are air-borne and are spread by wind, splashing rain and moving objects. As the disease develops rapidly in hot or humid conditions, it is likely to occur in July or early August. The fungus attacks younger leaves in preference to older ones, in contrast with alternaria blight.

CONTROL:

1. Practice a three-year rotation to prevent inoculum from surviving in crop refuse.

2. Treat the seed with a protective fungicide before planting.

3. If necessary, spray the foliage with a recommended fungicide such as chlorothalonil (Bravo), maneb (Zineb or Dithane M-22) or zineb (Dithane Z-78). Begin the treatments when the carrots are 10-15 cm tall and make two consecutive applications, 7-10 days apart. Then repeat the treatment according to weather conditions or to disease forecasts. It is preferable to begin the applications of fungicide early to prevent the buildup of inoculum. It is also easier to obtain better coverage and good protection when the foliage is not too dense. Preharvest intervals = 1 day (chlorothalonil), 5 days (maneb), 7 days (zineb).

4. Plant resistant varieties (see page 59).

ASTER YELLOWS

PATHOGEN: Aster Yellows Mycoplasma

SYMPTOMS: Carrot plants infected with aster yellows show a marked dwarfing, twisting and yellowing of the young central leaves, while outer older leaves remain normal in size although bronzed or tinged with red or purple. A dense growth of sticky yellow shoots may develop at the center of the crown. Numerous stunted hairlike rootlets commonly arise from the root. Affected carrots may be smaller with a bitter off flavor. Flower development may be aborted, producing only leaflike structures.

DISEASE CYCLE: The causal organism, a mycoplasma, is perpetuated in infected perennial crops and weeds, various plants growing in the greenhouse, in bulbs, corms, tubers and other propagative units. Perennial weeds that retain the organism from one season to another include thistle, plantain, wild chicory, wild lettuce, daisies and many others. The leafhopper is also an overwintering host in mild climate areas.

The chief vector is the aster leafhopper. Infected insects migrate northwards in spring and early summer, feeding and infecting new hosts. Young leafhoppers sucking juices from the newly infected hosts pick up the pathogen. After a short incubation period, these insects are capable of transmitting the mycoplasma to neighboring plants. Disease incidence is not always correlated with the population of the insect vector in the field. Important considerations are the relative abundance and kind of mycoplasma reservoir, temperature and other climatic conditions affecting the pathogen, host, vectors and leafhopper mobility.

CONTROL:

1. Do not plant susceptible crops adjacent to yellows-infected crops.

2. Where possible, eradicate all susceptible perennial and biennial weeds along roadways, ditch banks, fences and in pastures near carrot fields.

3. Frequent applications of insecticides will control leafhopper vectors during heavy migratory periods. Insect host weeds growing near flowers and vegetable crops should also be sprayed with insecticides to reduce vector sources. Frequent insecticide applications may be required during periods of migration.

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Bacterial Soft Rot of Carrots

General

Bacterial soft rot may occur in the field, but is more likely to occur in storage. Field infection is unlikely to become noticeable unless carrot roots have been subjected to particular stress caused by such things as excessive flooding, high temperatures, or mechanical injury including insect attacks.

The disease can be held in check at temperatures below 5°C. Washed prepackaged carrots must be kept at continuous low temperature in storage and transit. In non-refrigerated root cellars, the disease is most likely to occur toward the end of extended storage periods.

Signs and Symptoms

A distinguishing feature of the disease, as it affects carrots, is the presence of a glistening, watery, slimy covering of affected parts. Outer tissue of affected parts often tends to remain intact, while underneath, decayed portions are very soft and water-soaked. The causal organism reproduces very rapidly in carrots, and typical water-soaked symptoms usually appear 12 to 24 hours following infection. The core of the root appears to rot first, and a sharp line can be seen or felt between diseased inner and healthy outer tissues. Smell of the rot can be detected in carrots, but it does not produce the same foul odor as occurs in rutabagas and other cruciferous corps. Leaves of carrots becoming infected in the field may turn yellow or wilt and later die.

Causal Organism

The soft rot bacterium, Erwinia carotovora (Jones) Holland, is widely distributed and has a very extensive host range. Among the crop and weed plants attacked are rutabaga, turnip, parsnip, carrot, cabbage, cauliflower, tomato, lettuce, celery, potato, cucumber, squash, pumpkin and several species of cruciferous weeds. At high temperatures and high humidity, it is extremely virulent on carrots.

Control Measures

Crop Rotation: Owing to the wide host range of the soft rot bacterium, crop rotation is only partially effective as a control. However, the disease is less destructive when susceptible crops are rotated with non-susceptible crops like corn, cereals, flax, beans and grasses.

Cultivation and Irrigation: Since the causal organism thrives in overly-wet soils, it is desirable to avoid planting carrots in low, wet, cold soil Excessive irrigation, particularly of heavy soils, in hot weather, may result in plant stress and subsequent soft rot infection. During cultivation, it is desirable to avoid mechanical injury to carrot foliage, crowns, or secondary roots. Also, avoid frost injury to above-ground parts if possible. Sprinkler irrigation may encourage infection during warm weather, especially if growing plants have been damaged in the field

Insect Control: Several species of maggot flies may carry in their intestinal tract the soft rot bacterium, which covers their eggs laid in the soil near plants. Some types of insect larvae require the bacterium for normal development, and in turn aid in the spread of soft rot. Larvae may also become contaminated through contact with rotted plant debris in the soil. Control of some fly larvae such as the seed corn maggot may be justified, especially for crops growing under stress or subject to mechanical injury

Harvesting and Storage: Since soil particles adhering to newly harvested carrots are likely to contain the soft rot bacterium, every effort should be made to avoid bruising, cutting or cracking the tap root. Frequent washing of storage houses and equipment with a suitable bactericidal disinfectant is a useful precaution against possible contamination of healthy carrots. Likewise, bactericidal agents used in hydrocoolers or wash water can prevent a serious buildup of inoculum. To be effective, however, the concentration of such solutions must be checked and corrected repeatedly especially if considerable soil or foliage enters the water. Some bactericides are effective only in water that is somewhat acid or alkaline (pH measurement). Chlorine washes, to be effective, must be kept within pH range of 8.0 to 8.5 (slightly alkaline).

Germicidal washes are not a substitute for careful handling and storage procedures. In fact, almost any carrot after exposure for several minutes to liquid commercial bactericides can be found to still harbor disease organisms in tiny protected surface crevasses, depressions, cracks or splits. Also, germicidal washes have no beneficial effect whatsoever in preventing decay of carrots which have already been penetrated by decay organisms.

The most effective soft rot prevention in storage and transit is careful temperature control. Healthy carrots, when placed in bins at temperatures consistently kept below 5°C are not likely to be attacked by the soft rot bacterium unless the roots have been dug too early, touched by frost or injured during handling. Even damaged carrots will seldom be affected by soft rot when they are stored at 0 to 2°C.

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Cottony Soft Rot of Carrots

General

Cottony soft rot is a fungus disease which probably accounts for greater storage losses in Alberta than do all other carrot storage diseases combined. Although it can affect carrots in the field, its presence is most commonly detected in storage. Losses as high as 30 to 50 per cent are not uncommon in carrots stored 3 months or more. The disease may completely destroy an entire stored crop.

Signs and Symptoms

The most noticeable sign of the disease is the presence of abundant cottony white mycelium often accompanied by small, hard, black bodies (technically known as sclerotia and considered to be resting bodies). Affected roots develop a slight discoloration in the infected area and a watery soft rot and "leakage." Infection may occur anywhere along the length of a carrot root, although it is more likely to begin near the top.

Causal Organism

The causal fungus Sclerotinia sclerotiorum (Lib.) DeBary is world-wide in occurrence, except in warmer areas of the tropics. In addition to infective mycelium and sclerotia already mentioned, air-borne spores are likely produced in Alberta fields under excessive warm, moist conditions. One of the most damaging features of the organism is its capacity to develop at low temperatures. High humidity, such as is required for quality carrot storage, favors growth and developlent of the fungus. Once inside the root its moisture requirements are readily met by cell "leakage." Direct tissue penetration and disease development are made possible by the cell-dissolving enzymes and toxins secreted by the fungus.

Control Measures

Crop Rotation: Careful crop rotation and weed control are important measures for reducing the amount of fungus inoculum in the soil. In view of the fact that numerous crops and weeds are susceptible to attack (for example, peas, beans, lettuce, sunflowers, parsnips, beets, turnips, cruciferous weeds and others), effective control depends upon rotating susceptible crops by groups and on thorough weed control. A minimum of 2 years (and preferably 4) of sclerotinia-resistant crops is necessary to assist the beneficial soil micro-organisms is cleaning up infested soil. Potatoes, onions, cereals and grasses are useful sclerotinia-resistant crops to include in a preventive crop rotation. Control of broad-leaved weeds such as mustard, stinkweed and shepherd's — purse will increase the efficiency of crop rotation aimed at reducing the amount of fungus inoculum in the soil.

Sclerotia are not killed by outdoor freezing and may persist for 2 or more years in infested soil. They are decomposed, in time, by beneficial micro-organisms particularly under warm wet soil conditions.

Storage Temperature: Cold carrots tend to store better than warm carrots, so digging in late fall when the soil is cooler is a distinct advantage. Once the carrots are cooled, 0 to 2°/C it is of utmost importance that uniform low storage temperatures be maintained. Rises in temperature to only 3 to 5°C for a few hours are sufficient to enable the fungus to penetrate healthy carrots. The fact that "pockets" of sclerotinia-infected carrots frequently occur within piles or pallets indicates that uniform cold temperatures are not being maintained throughout. If left undisturbed, such pockets will shortly begin to "leak" and nearby healthy carrots will quickly become diseased. Maintenance of low storage temperature is required to prevent serious losses. Affected carrots should be culled out whenever possible. Where large quantities of produce are being stored, "pockets" or "nests" of sclerotinia-infected carrots may remain undetected for weeks until "leakage" becomes noticeable. At this time, not only carrots that are obviously infected but "wet" ones also should be immediately removed.

Sanitation: Field and storage sanitation measures are important in preventing spread of the fungus responsible for cottony soft rot. Sclerotia may remain alive for months in cracks or crevices of handling equipment or storages, and may serve as a source of infection in subsequent susceptible stored crops. Pressurized steam treatment is useful in killing sclerotia, whereas chemical treatments are ineffective. Cull carrots should be steam-treated or boiled in order to prevent spread of the disease-causing fungus to fields where carrots or other sclerotinia-susceptible crops may be grown. This precaution is especially important where cull carrots are being used to feed livestock.

Communications Branch — 9718 - 107th Street, Edmonton, Alberta T5K 2C8

RUBBERY ROOT

PATHOGEN: *Phytophthora porri*

SYMPTOMS: In the early stages, affected plant tissues assume a water-soaked appearance. Later, the tissues turn brown but remain firm and watery. Affected roots may have a rubbery texture. In storage, white cottony growth of the fungus frequently covers the surfaces of roots. This symptom is similar to that of white mold caused by *Sclerotinia sclerotiorum*, but no black sclerotial bodies are formed by *Phytophthora*. A sweet odor may accompany this storage decay.

DISEASE CYCLE: The fungus survives in soil and appears to be favored by high moisture levels. Infected cull roots and plant debris in the fields are a source of contamination.

CONTROL:

1. Rotate carrots with cereals or grasses.
2. Provide adequate soil drainage and avoid excessive irrigation.
3. Maintain 0-2°C temperatures and adequate ventilation in storage.
4. Disinfest contaminated storages with steam or chemicals.

CELERY

ASTER YELLOWS

See CARROTS - Aster Yellows

BLACKHEART

CAUSE: A physiological disorder that also affects less severely parsley, carrots, and other umbelliferous crops. The disease is also related to lettuce tipburn, pepper seedling tipburn, cabbage tipburn and tomato blossom end-rot.

SYMPTOMS: The first symptom, which usually does not appear until the plant is relatively mature, is a water-soaking of the tips of the youngest leaves in the center of the celery plant. These areas quickly turn brown and enlarge until all the inner heart leaves die. Outer stalks are never affected. Usually, soft-rot bacteria follow and cause a watery, slimy decay.

DISEASE DEVELOPMENT: Blackheart can become a problem when the following conditions coincide: hot weather, rapid plant growth and soils high in fertilizer salts. It tends to be more common in low spots in fields. Symptoms are usually seen following a series of cloudy days and relatively high temperatures, especially at night, with soil moisture optimum for plant growth. The low light conditions permit a greater amount of guttation water containing soluble salts to exude through the cell membranes and collect on the surface of the plant, especially along the youngest, most rapidly growing parts. As the guttation water dries, the salts become concentrated enough to cause killing of the tissues. When sodium, phosphorus and magnesium are present, permeability increases resulting in a greater liberation of soiuble salts. If calcium is in deficit, the disorder is greatly increased as calcium reduces membrane permeability to soluble salts.

CONTROL:

1. When starting plants in the greenhouse, keep the humidity and night temperatures low.
2. Plant in well-drained soil.
3. Water during dry periods to maintain uniform soil moisture.

4. Avoid over fertilizing with high salt index fertilizers.

5. Apply sprays of calcium chloride using 0.5 - 1.0 kg/100 L of water or calcium nitrate at 1.0 - 2.0 kg/100 L into the heart leaves at weekly intervals.

6. Harvest promptly at maturity.

CORN

BACTERIAL STALK ROT

PATHOGEN: *Erwinia* sp.

SYMPTOMS: First symptoms generally occur in midseason when the tops of the plants wilt and turn a straw brown color. The leaves eventually dry up. If stems are cut in cross section the inseide appears brown and decomposed. Often, there is a disagreeable odor associated with the rotted tissues. The decay may advance down the stalk, eventually causing death of the outer leaves.

DISEASE CYCLE: Bacteria appear to invade the plant through the top whorl of leaves. Thus far in Alberta the disease has only been found on corn under pivot irrigation. Bacterial stalk rot appears to be favored by high temperatures. The origin of the bacteria is uncertain, however, it appears they may be carried in the water applied to the crop.

CONTROL:

1. Avoid overhead irrigation with stagnant water from holding ponds, ditches, etc.
2. Rogue infected plants, if practical.

COMMON SMUT

PATHOGEN: *Ustilago maydis*

SYMPTOMS: The main symptom is the formation of conspicuous swellings (galls). All above-ground parts of the plant are susceptible, particularly young, actively growing tissues. Galls are at first covered with a glistening, greenish to silvery-white tissue. The interior of these galls darkens and turns into masses of powdery, dark olive-brown to black spores, except for galls on leaves. As galls mature, they may reach 15 cm in diameter. Galls on leaves usually remain small, 0.6-1.2 cm in diameter, become hard and dry out but do not rupture. Early infection may kill young plants, but this is rare. Plants with galls on lower stalks may be barren or produce several small ears.

DISEASE CYCLE: Resistant thick-walled spores overwinter and germinate under favorable conditions to produce other smaller spores which are carried by air currents or splashed by water to young developing corn plants. Strands of fungal growth from the germinating spores penetrate the plant through breathing pores (stomata) on the leaf surface, through wounds or directly through cell walls stimulating host bells to proliferate. Smut development is favored by dry conditions and temperatures between 26 and 34°C. The interval between infection and gall formation varies from one to several weeks under favorable conditions. Disease incidence is generally higher among plants grown in soils high in nitrogen or after heavy manure application. Injuries due to hail, blowing soil particles or cultivation greatly increase the potential of smut infection.

CONTROL:

1. Avoid planting susceptible varieties. Calico is resistant to smut.
2. Avoid injuring plants through cultivation or other methods.
3. Maintain balanced soil fertility. In particular, avoid high nitrogen levels.
4. Where practical, remove and destroy galls by hand as soon as they appear.

HOLCUS LEAF SPOT

PATHOGEN: *Pseudomonas syringae*

SYMPTOMS: Circular to oval spots ranging from 2-10 mm in diameter appear over the leaf surface. Generally, the spots first occur on lower leaves and are concentrated towards the edges and tips. At first, the spots are dark green and water-soaked, later they turn creamy-white to tan in color. Larger lesions may be surrounded by a yellowish halo. Leaf tips may turn completely brown and appear scorched. A crop severely affected by holcus leaf spot appears as if it has been injured by frost. Leaf tissues are weakened by the lesioning and often shred in the wind.

DISEASE CYCLE: The bacteria may be carried as contaminants with corn seed. It may also be possible that the organism overwinters in crop residues under Alberta conditions; however, there is no direct evidence of this to date. *P. syringae* has been observed to attack the leaves of several grasses,including wild oats, barnyard grass, wild millet and barley in corn fields in southern Alberta. Warm, rainy, windy weather, especially early in the season, appears to favor disease development.

CONTROL:

1. Follow a two-year crop rotation with broadleaved special crops. Avoid sorghum, millet, sudan grass and susceptible cereals.

2. Maintain adequate potassium levels in the soil.

SEED ROT AND SEEDLING BLIGHT

PATHOGEN: Various soil and seed-borne fungi, e.g., *Pythium* spp., *Fusarium* spp., *Rhizoctonia solani*, *Penicillium* spp.

SYMPTOMS: Above-ground symptoms of these diseases are yellowing, wilting and death of leaves. They may be confused with chemical or mechanical injury or insect damage. Examination of parts in the ground is therefore necessary for accurate diagnosis. Following are descriptions of the various phases of seed rot and seedling blight diseases:

a) Seed rot - rotting of seed before germination
b) Damping-off and seedling blight - soft rot of stem tissues near ground level and water-soaking of seedling tissues.
c) Root rot - water-soaking, browning and sloughing of rootlets. The rot may advance into main roots and seedling or crown tissues.

DISEASE CYCLE: Strands of fungus growth (mycelium) contact seed or seedling tissue and enter the seed through cracks in the seed coat or by direct penetration. The mycelium grows rapidly through and between the cells, killing the seed. Similar attack may occur through rootlets and stems by direct penetration or through wounds. The mycelium proliferates in young cells causing rapid collapse and death of tissues.

CONTROL:

1. Slow injury-free seed or resistant varieties (consult seed suppliers about disease resistance of their varieties).

2. Follow good cultural practices, e.g., plant in warm, fairly moist soil, properly prepare the seed bed, correctly place fertilizer relative to the seed, and use fertilizer and herbicides to insure maximum seedling vigor.

3. Apply seed protectant fungicides, e.g., Captan, Thiram, or Vitavax-Thiram. Most of these are available as dual purpose formulations with lindane insecticide for wireworm control.

LETTUCE

BACTERIAL SLIME ROT

PATHOGENS: *Pseudomonas* spp. and *Xanthomonas* spp.

SYMPTOMS: The disease shows as a leaf spot or as a stem rot. The leaf spots usually begin as V- or irregular-shaped areas at or near the margins. Spots sometimes enlarge until half or more of the leaf is included. As the spot enlarges, the center darkens with a wilted olive or brown colored margin. Although the dead area appears moist it does not become soft and when dry is papery in texture. In continued wet weather the bacteria occasionally ooze to the surface, causing a slight yellowish sheen on the blanched head leaves. In a cross-section of the stem there is a discolored area that may enlarge until the whole stem is rotted, especially in young plants. The injury in the stem is accompanied by progressive stages of wilting or by a stunting.

DISEASE CYCLE: Little is known about the survival and spread of the causal bacteria under Alberta conditions. It appears likely that they are spread by splashing water, insects and on transplanted seedlings. Survival may occur on infested debris which is left in the field.

CONTROL:

1. Avoid overcrowding and overwatering.
2. Harvest promptly when mature and pre-cool at 1°C immediately.
3. Control pest insects if present.
4. Avoid unnecessary injury to growing plants and harvested heads.
5. Practice crop rotation.

FUNGAL ROTS

PATHOGENS: *Botrytis cinerea, Rhizoctonia solani* and *Sclerotinia sclerotiorum*

SYMPTOMS: Botrytis Gray Mold - Older leaves, especially those which may be touching the soil, are usually the first tissues attacked. Other common sites of infection are leaf margins and the center of the head of compact-headed lettuce varieties. Infected areas rapidly turn brown and soon may become covered with felty masses of gray-brown spores of the causal fungus. Severely infected heads may rot completely.

Rhizoctonia Canker - Young plants are usually the hardest hit. Pre- and postemergence damping-off may occur if conditions are favorable. Tissues invaded by the *Rhizoctonia* fungus are usually brown colored and constricted.

Sclerotinia Rot - The causal fungus can attack lettuce plants at any stage of growth. On younger plants, symptoms of this disease may include rapid wilting and death. On older plants, the fungus invades the main stem at or near ground level. A water-soaked area develops which may spread downwards until the roots are decayed. The leaves turn yellow, wilt and collapse. Under humid conditions, a white cottony mass of fungal growth grows up from the watery brown decayed tissues. Dark black resting bodies (sclerotia) eventually may form on these areas.

DISEASE CYCLE: All three fungi are soil borne; however, under favorable conditions, airborne spores may be produced which enhance spread.

CONTROL:

1. Turn under crop refuse promptly after harvest.
2. Avoid poorly drained fields and overcrowded planting.
3. Discard heads with any trace of infection at harvest.
4. Pre-cool promptly after harvest.

TIPBURN

CAUSE: This is a nonparasitic disease which appears to be most prevalent when bright warm days follow periods of damp foggy weather. Several factors have been reported to influence the amount and severity of the disease:

1) rapidly growing, succulent plants tend to be more susceptible than plants that have developed more slowly
2) high humidity and high temperatures favor tipburn
3) fluctuating or inadequate soil moisture may increase the amount of tipburn
4) the disease may be related to calcium and/or boron deficiencies.

SYMPTOMS: The first symptoms are small, brown spots about 0.6 cm from the edge of the leaf. The tissue around these spots often dies and turns brown. Tipburn may also occur internally; necrotic spots and patches of veinlet necrosis near leaf margins may appear throughout the interior of the head. Such dead areas within the head offer excellent places for rot-producing organisms to become established, especially the bacteria that cause a watery tissue breakdown commonly known as "slime rot".

CONTROL:

1. Use varieties which show some resistance, e.g., Alaska Great Lakes, Salad Bowl, etc.

2. The control measures for celery black heart also should reduce the incidence of tipburn.

ONIONS

BASAL ROT

PATHOGEN: *Fusarium oxysporum* f. sp. *cepae*

SYMPTOMS: Yellowing and drying of leaves from the tips as the plants are approaching maturity are the first symptoms of basal rot. The roots of affected plants may eventually rot off and a pinkish-white mass of moldy growth may be produced. Rot may develop at the base of the bulb and eventually progress into the scales causing a semi-watery decay. Bulbs may decompose in the field or in storage, especially when storage temperatures exceed 4°C.

DISEASE CYCLE: The *Fusarium* fungus survives in soil and invades onion roots and bulbs through wounds caused by onion maggots or other agents. Diseased onion sets are also a source of infection.

CONTROL;

1. Follow a four-year crop rotation.
2. Avoid close cultivation to prevent bulb and root injury.
3. Practice root maggot control where needed.
4. Cure and store bulbs at the proper temperatures.
5. Grow varieties with some resistance to the disease, e.g., White Portugal, Stokes Explorer and the yellow globe types.
6. Plant disease-free onion sets.

NECK ROT

PATHOGENS: *Botrytis allii* and other *Botrytis* species

SYMPTOMS: Neck rot is principally a storage disease and seldom attacks onion bulbs while they are still in the ground. The tissue about the neck of the onion may become sunken and have a dried out appearance. A gray mold with many black kernel-like bodies (sclerotia) may appear about the neck of the onion or cover most of the surface. Inner scales are soft, appear as if they have been cooked and have a light-brownish color. Between the scales, the mycelium of the fungus may be noted in gray masses. When soft rot bactertia follow neck rot the entire onion is reduced to a soft, wet, foul-smelling mass.

DISEASE CYCLE: *Botrytis* overwinters as sclerotia in trash, soil and buildings that have been used to store diseased onions.

CONTROL:

1. Use early maturing varieties which have small, tight necks.
2. Seed as early as possible in the spring.
3. Avoid harvesting or handling onions in wet weather.
4. Thoroughly cure onions at room temperatures of 32-35°C for one to two weeks under continuous air movement. Thereafter, reduce temperatures to 0-4°C and provide less than 70% relative humidity.
5. Practice crop rotation by avoiding growing onions for two years in succession on the same piece of land.
6. Prevent late growth by avoiding heavy applications of nitrogen.
7. Colored-bulb varieties are less susceptible.

SMUT

PATHOGEN: *Urocystis cepulae*

SYMPTOMS: This fungus attacks onions and leeks, but not garlic. The first symptoms appear as dark streaks in cotyledons of seedlings. Similar symptoms appear on other leaves as they develop. Affected leaves soon swell and are distorted. Infected plants may survive through the summer but usually die within one month after seedlings emerge. Blisters in localized areas on leaves and bulbs often rupture to expose black powdery spore masses. Smutted bulbs are more susceptible to rotting in fields and storages.

DISEASE CYCLE: The fungus remains viable for an indefinite number of years in the soil and is spread with soil. The smut of onions is confined to areas the cool summer temperatures.

CONTROL:

1. In areas where smut does not occur, avoid introduction of transplants or sets from areas where the disease does occur.
2. Clean equipment thoroughly when moving from smut-infested fields to new fields.
3. In heavily infested fields, transplants can be set out safely as only the seedling stage is susceptible to infection.
4. If there is a chance that smut is present in a field, consider planting when the soil temperature is high (approximately 21°C) as high temperatures inhibit fungal spore germination.
5. Coat the seed with thiram or thiram-carbathiin (ProGro). Thiram may also be used as an in-furrow treatment. The efficacy of thiram + carbathiin seed treatment is improved sugnificantly by the use of methyl cellulose as a seed sticker.
6. Formaldehyde can be applied to the soil at the time of seeding for smut control. It volatilizes sufficiently to inhibit the fungus for the susceptible period of the seedlings. Mix 7-14 L of 37% formaldehyde in 1000-2000 L of water per hectare. Caution: Injury may occur in dry soils.
7. There are no resistant varieties of bulb onions, but Beltsville Bunching and Nebraska green bunching onions have considerable resistance.

WHITE ROT

PATHOGEN: *Sclerotium cepivorum*

SYMPTOMS: The fungus infects onion, Welsh onion, garlic, leek, shallot and some species of wild onion. The trouble is found mostly in the field, seldom causing injury in storage. The number of plants that show infection depends upon the amount of inoculum in the soil. Diseased plants are nearly always scattered about the field and seldom comprise a continuous part of any row. The leaves of an invaded plant decay at the base, turn yellow, wilt and fall over, the older ones being the first to collapse. The roots of such plants usually are badly rotted, so that the plant can be pulled up easily. A white superficial fluffy mold develops on and in affected bulbs. Numerous sclerotia (black spores) about .5mm in diameter are formed throughout the infected parts and on the surface of the onion. They are not as large or as nearly flat as are those of *Botrytis*.

DISEASE CYCLE: It is not known how long the fungus can persist in the soil in Alberta without the presence of the host. In some other onion-growing areas, it has survived for 8-10 years. This persistence probably is due to the numerous sclerotia, which lie dormant when moisture and a host are present, germinate and within a few days cause infection in onions at any stage of their growth. Within a week of infection more sclerotia are produced. The fungus also lives over in slightly diseased sets, in wild onions, or as sclerotia mixed with the seed. In addition, it is also spread in seedlings that have not been infected long enough to show symptoms before being planted. The pathogen can be washed from field to field by running water or carried in soil clinging to transported seedlings or machinery. The disease is most severe in cool, moist soil.

CONTROL:

1. In areas where the disease does not occur, do not introduce transplants or sets from areas where it does occur.

2. To prevent field-to-field and farm-to-farm spread, clean equipment after leaving an infested field before entering a clean field.

3. Dispose of infected culls in such a way that they will not be returned to fields where onions are to be grown.

4. Avoid seeding onions in fields where white rot occurred that last time onions were grown.

5. Take steps to prevent movement of flood waters from infested to clean fields.

6. If onions must be grown in infested soil, the following treatments may reduce losses: for seeded onions on mineral soils, broadcast dichloran (Botran 75 WP) at 30 kg/ha and rotovate to a depth of 4 cm, 1-2 weeks before seeding; for transplants, apply dichloran at 15 kg/ha in the furrow at planting.

PARSNIPS

ASTER YELLOWS

See CARROTS - Aster Yellows

SCLEROTINIA ROT

See CARROTS - Cottony Soft Rot

PEAS

POWDERY MILDEW

PATHOGEN: *Erysiphe polygoni*

SYMPTOMS: Mildew is characterized by a white powdery or talcum-like mold on the leaves, stems and pods. Later in the season, the white mold may be interspersed by numerous small black spots about the size of a pin head. These are fruiting bodies called cleistothecia. Usually, severely infected plants wither and on occasion the plant may die. Sometimes the fungus grows through the pea into the seeds so that they adhere to the pod. Seeds may be discolored and pods which are late in developing may contain no seeds where infection is severe.

DISEASE CYCLE: The disease is seed borne and is spread through infected seed. It also overwinters on crop residues.

CONTROL:

1. Use disease-free seed.
2. Plow down diseased crop residues.
3. Application of finely ground sulphur as a dust will help retard the disease. Apply as soon as symptoms appear and thereafter at weekly intervals for about four weeks. Preharvest interval = 1 day.
4. Early seeding allows the plants to reach a more advanced stage of development before infection, hence less effect will occur on yield.

ROOT ROT

PATHOGENS: Several fungi: *Fuasrium solani* f. sp. *pisi*, *Pythium* spp. and *Rhizoctonia solani*.

SYMPTOMS: Roots and lower stems develop to black lesions, often beginning at the point of seed attachment. When the disease is severe the below ground parts of the plant are dark brown and rotted and the lower stem is constricted below the soil surface by collapse of rotted tissue. Severely affected plants are stunted, become pale or yellow, lose their lower leaves and die prematurely.

DISEASE CYCLE: The pea root rot fungi are soil-borne. Populations gradually decrease in soils not cropped for several years to susceptible hosts, but increase rapidly in soils cropped to peas and certain other legumes.

CONTROL:

1. Plant peas no more frequently than once in five years on the same land.
2. Avoid green beans, fababeans and alfalfa in rotations containing peas.
3. Use of a recommended seed treatment fungicide, i.e., Captan or Thiram, will assure an adequate stand but will not control root rot.

SEED ROT AND DAMPING-OFF

PATHOGENS: *Pythium* spp.

SYMPTOMS: Seedlings fail to emerge or become stunted and collapse before the first leaves unfold. Seeds rot rapidly and become encased in a white moldy growth intertwined with soil particles. Plants that escape seedling damage sometimes develop a blight of the growing point at a leaf axil several centimetres above ground. The tissue becomes water-soaked, rapidly dies, and turns brown or greenish-purple and shrivels. New shoots often develop from a leaf axil below the damaged area.

DISEASE CYCLE: High populations of these fungi occur as dormant bodies in most cultivated soils. Infection, which is triggered by sugars and other nutrients released from germinating seeds, occurs within a few hours in wet soils. Damage is favored by heavy rain or irrigation shortly after the crop is seeded.

CONTROL:

1. Seed into a moderately firm seedbed.
2. Rotate peas with grain crops.
3. Treat seed with a recommended fungicide, e.g., Captan or Thiram.

POTATOES

Detailed descriptions of the common diseases of potatoes in Alberta and measures for their control are discussed in A.H.R.C. Pamphlet #83-14 entitled "Diseases of Potatoes in Alberta". Copies of this publication can be obtained by writing to:

The Library
Alberta Horticultural Research Center
Bag Service 200
Brooks, Alberta
T0J 0J0

RADISH

DAMPING-OFF

PATHOGENS: *Pythium* spp. and *Rhizoctonia solani*

SYMPTOMS: Pre-emergence damping-off - Seed rots in soil or seedling dies before it comes up.

post-emergence damping-off - Seedlings affected after emerging from soil but while still small and succulent. Stem tissues at the soil line become water soaked and browned or blackened depending on the fungus involved. Shrivelling of affected tissue may follow and seedlings topple over.

CONTROL:

1. Plant in a well-prepared, well-drained seedbed.
2. Treat seed with Captan or Thiram prior to planting.
3. Do not sow seeds too thickly.
4. Avoid overfertilizing especially with nitrogen.

RHUBARB

CROWN ROT AND RED LEAF

PATHOGENS: *Erwinia rhapontici* and Turnip Mosaic Virus

SYMPTOMS: *E. rhapontici* incites crown rot disease of rhubarb. The main symptoms are rotting of the terminal bud, a soft chocolate colored rotting of the pith and the formation of a cavity. Spindly side shoots may grow out but these usually rot off. During wet weather, the bases of older leaves may become affected. Dull red colored leaves are usually present on the diseased plants. Turnip Mosaic Virus causes dwarfing, mottling and reddening of the leaves.

DISEASE CYCLE: Relatively little is known about the spread and survival of the crown rot/red leaf pathogens in Alberta. Transplanting of infected crowns, and foliage- or root-feeding insects appear to be most likely methods by which the diseases are spread.

CONTROL:

1. Obtain disease-free crowns for planting.
2. Rogue out plants with obvious symptoms of red leaf or crown rot.
3. Do not allow populations of aphids or other foliage-feeding insects to build up on rhubarb plants.

RUTABAGA

BLACKLEG AND BLACK ROT

PATHOGENS: *Phoma lingam* and *Xanthomonas campestris*

SYMPTOMS: See COLE CROPS: Blackleg and Black Rot

DISEASE CYCLE: See COLE CROPS: Blackleg and Black Rot

CONTROL:

1. Use hot-water-treated seed (20 min at 50°C) for blackleg.
2. Follow a four to five year rotation with non-cruciferous crops for blackleg or a 2 year rotation for black rot
3. Do not apply compost containing rutabaga refuse or diseased rutabagas to land intended for crucifer production.
4. Avoid storing diseased roots.
5. Clean and disinfest storages each spring.

SOFT ROT

See CARROTS - Bacterial Soft Rot

WATER CORE (BROWN HEART)

CAUSE: Boron deficiency

SYMPTOMS: The symptoms in rutabagas that draws most attention is glassiness or water-core in the flesh of the edible root. This condition does not show on the surface except occasionally as a roughening or russeting of the skin. The water-soaked tissue gradually turns gray and finally brown or black and becomes punky in texture. The leaf symptoms may be lacking or dead. Cross-hatched areas may appear on the inner surface of leaf stalks and young, unfolding leaves turn black and die.

Turnips exhibit the water-soaked tissue only briefly but have internal gray, punky flecks or spots that later change to brown or black areas. Otherwise, the symptoms resemble those in rutabaga.

CONTROL: In light, sandy soils or where boron deficiency has occured in the past, broadcast and disc in agricultural borax (11.4%) at 40 kg/ha or Tronabor (14.0%) at 30 kg/ha before planting. If deficiency occurs after planting, immediately apply Solubor (20.5%) or Borospray (20.5%) at 0.55 to 1.1 kg/ha as a 0.1% spray, using at least 1120 litres of water/ha.

Fields to which boron has been applied ahould not be planted to beans or cucumbers the following year as both these crops are extremely sensitive to boron.

WIRESTEM AND DAMPING-OFF

See COLE CROPS - Wirestem and Damping-Off

TOMATOES, PEPPERS AND EGGPLANTS

BACTERIAL BLIGHTS (Tomatoes)

PATHOGENS: Bacterial canker (*Corynebacterium michiganense*);
Bacterial spot *(Xanthomonas vesicatoria)*
Bacterial speck *(Pseudomonas tomato)*.

SYMPTOMS: Bacterial canker - Occasionally, seedlings are infected, but often they show no prominent symptoms until several days after they have been transplanted to the field. Wilting of the leaflets is the first symptom on plants of all sizes. On older plants the margins of the leaflets wilt first. The affected leaflets become brown, and they die progressively from the margins in toward the midrib. Usually the leaflets on one side of the leaf are affected first. The plant often shows a one-sided development of the disease. Infected plants may wilt and die early, if the growth is soft. However, if the growth is hard, many of the affected plants do not die, but survive in an unthrifty, wilted condition and bear some fruit. During the hot summer months, infected plants show the greatest capacity to survive.

Light-colored streaks extending up and down the outside of the stem and on the petiole may accompany wilting of the leaves. When these streaks occur, they sometimes break open to form a canker. As decay progresses, the pith becomes yellow and mealy, and cavities form in the soft tissues of the stem. When an infected stem is gently pressed between the thumb and forefinger at the nodes, a sponginess is detected. When the stem is cut lengthwise, there is a creamy white, yellow or tan streak just outside the woody tissue and a softening of these tissues develops.

If crops are watered by sprinkler irrigation, small bird's-eye cankers may appear on the surface of fruits on infected plants. These infections appear first as snow-white spots that extend scarcely beyond the skin. The margins of the spots remain white and flat; the centers become slightly raised, tan colored and crack open. These spots do not exceed 3 mm in diameter. The halo around the spot is an aid in distinguishing canker from the bacterial spot disease. The bird's-eye spot is a common symptom of the disease in field crops.

Bacterial spot - Bacterial spot causes spotting on leaves, stems and fruit. The most conspicuous spots are on the fruit. On the leaves and stems, small (1.5 mm) black spots with a greasy appearance may develop. On the fruit, the spots form slightly raised, rough, scabby, dark-colored lesions, in the center of which numerous cracks may occur. Spots may enlarge to 6 mm in diameter. They do not extend deep into the fruit.

Bacterial speck - It is most noticeable on the fruit, where it causes numerous tiny dark brown specks less than 1.5 mm in diameter. These spots do not extend much deeper than the thickness of the skin. Because both bacterial speck and bacterial spot may occur on the same fruit, the larger speck lesions are often mistaken for small lesions of bacterial spot. On the leaves, bacterial speck causes dark brown to black spots that are usually slightly larger than those on the fruits.

DISEASE CYCLE: Bacterial canker - Contaminated seed is often the source of primary infection. It can also be carried on infected crop debris. In stalked field crops, the disease is readily spread from plant to plant by pruning, tying and handling. In processing and basket crops, canker is largely spread by rain and irrigation. Under field conditions in Alberta the bacteria are unlikely to survive more than a year.

Bacterial spot - the causal bacterium is seed-borne. The disease spreads during heavy rains. The causal bacteria also attack peppers and produce lesions on the fruits and leaves similar to those on tomato.

Bacterial speck - The causal bacterium is seed-borne. Infection occurs most abundantly after heavy rains that splash the bacteria to all parts of the plant. This disease does not infect peppers.

CONTROL:

1. Use only hot water-treated seed (50°C for 25 min) or disease-free transplants.

2. Grow seedlings in steamed or fumigated media in disinfested flats or pots.

3. If any of the diseases appear in a crop, carefully remove the first infected plants, avoid methods of pruning, etc. that may spread disease from plant to plant and avoid sprinkler irrigation.

4. Do not replant tomatoes or related plants for at least two years in a field where disease appeared.

5. If necessary, apply protective sprays of cupric hydroxide (Kocide 101 or Coppercide), cupric hydroxide + maneb or cupric hydroxide + mancozeb. Preharvest intervals = 1 day (cupric hydroxide), 3 day (cupric hydroxide + maneb), 5 days (cupric hydroxide + mancozeb).

BLOSSOM-END ROT (Tomato and Pepper)

CAUSE: An irregular water supply produces a water deficit in the fruit. Vigorous growth of tops followed by lack of water causes the leaves and stems to draw water from the fruit. Insufficient water uptake by roots due to poor root development, lack of water excessive transpiration due to wind and high air temperatures will also result in an irregular water supply and fruit moisture loss. Blossom-end rot is also associated with an unbalanced nutrient supply, particularly deficient calcium or excess nitrogen in relation to phosphorus, potassium and the other major nutrients.

SYMPTOMS: A slight water-soaked area forms at or near the bottom of the fruit (blossom-end), that later enlarges and darkens. Eventually, the cankers become dry, sunken, flat, leathery and finally dark brown to black colored in tomato or light-colored and papery in peppers. Various secondary micro-organisms may invade the injured tissues and fruit rots may follow.

CONTROL:

1. Water during dry periods to maintain even soil moisture supply.
2. Mulch or cultivate shallowly during dry periods to conserve soil moisture.
3. Fertilize adequately but avoid over-fertilization.
4. Avoid severe pruning and too close planting.
5. Foliar sprays of calcium chloride (500 g/100 L water) applied during the period of fruit development help to prevent blossom-end rot. Calcium nitrate (15.5-0-0) may also be applied at the rate of 100 g/100 L of water. A small amount of spreader-sticker should be added to the fertilizer solution to enhance distribution on the leaf surface.

EARLY BLIGHT

PATHOGEN: *Alternaria solani*

SYMPTOMS: The first symptoms of early blight usually seen are circular dark brown to black spots of various sizes on the leaves. As the spots enlarge, concentric ridges ('target spots') often form on their upper surface. On the stalks, branches and fruit pedicels, early blight appears as black lesions that subsequently enlarge and elongate and sometimes girdle the infected plant parts. On the fruit, infection results in large, black, leathery, sunken lesions. Under humid conditions, spores of the causal fungus may form on lesion surfaces.

DISEASE CYCLE: The fungus overwinters in old foliage, refuse, vines and leaves. High humidity, or water on the leaf surface, is necessary for infection and development of the disease. Sprinkler irrigation provides this necessary environment. During each sprinkler irrigation, a life cycle of the fungus can be completed. Wind is the chief means of disseminating the spores from leaf to leaf, plant to plant and field to field.

CONTROL:

1. Plow under crop debris immediately after harvest.
2. Avoid prolonged periods of high humidity if possible.
3. Follow a crop rotation that includes cereals and legumes.
4. A regular spray program at 7-14 day intervals may be required in commercial tomato fields from early June for control of early blight. Apply one of the following fungicides: chlorothalonil (Bravo), captafol (Difolatan), mancozeb (Dithane M-45 or Manzate 200). Preharvest interval = 1 day for all chemicals.

SUNSCALD

CAUSE: High temperatures and exposure of the fruit to intense, direct sunlight.

SYMPTOMS: Can appear at any place on the fruit. A light-colored area develops on the side of the fruit exposed to the sun. These areas become slightly sunken or wrinkled and creamy white on older fruit. Spots appear light brown in color on young fruit. Scalded tissues dry out rapidly in hot weather and become thin and papery. Growth of secondary fungi can result in black, dark green or gray mold on scalded surfaces.

CONTROL:

1. Varieties producing luxuriant foliage or fruit which hangs downward are less apt to be damaged by sunscald than those with upright fruit not well protected from the sun by the leaves.

2. Diseases causing loss of leaves may also contribute to damage from sunscald. These should be controlled if possible.

WILT

PATHOGENS: *Fusarium oxysporum* f. *lycopersici* and *Verticillium albo-atrum*

SYMPTOMS: With Verticillium wilt, the first foliar symptom usually observed is the wilting of one or more leaflets on a single leaf. The oldest leaves usually show symptoms first and then wilt develops progressively on the younger leaves. Frequently, the first symptoms appear on leaflets only on one side of the leaf. V-shaped yellowed areas extending in from the leaf margins are occasionally seen. A brown discoloration of the water-conducting tissues of the stem may develop in plants with well-established infection. Fusarium wilt causes a progressive wilting and death of tomatoes and usually affects plants more uniformly and drastically than Verticillium wilt. The vascular and woody tissues of the stems affected plants often show a brown discoloration.

DISEASE CYCLE: Both organisms are soil-borne and attack through the roots.

CONTROL:

1. In areas where any of these diseases do not occur, avoid introducing seedlings grown in areas where the diseases do occur.

2. When growing seedlings for transplanting, use steamed or fumigated media and disinfest flats and pots.

3. Follow a rotation in which members of the tomato-potato-pepper-egg plant group are not grown for at least 2 or 3 years.

4. Most commercial cultivars have resistance to *Fusarium* (F) and/or *Verticillium* (V). (See page 60).

VINE CROPS (CUCUMBER, MELONS, PUMPKINS AND SQUASH)

ANGULAR LEAF SPOT

PATHOGEN: *Pseudomonas lachrymans*

SYMPTOMS: Angular leaf spot is a bacterial disease which attacks cucumber leaves, stems and fruit. Spots on the foliage are straw-colored to light brown and angular shaped. Affected areas are first water soaked, then gradually dry and split. After the diseases tissue splits open, portions of it tear out, leaving irregularly shaped holes in the leaves. Small, circular spots develop on the fruit. These diseased areas later crack open and turn white.

DISEASE CYCLE: The bacterium which causes angular leafspot overwinters on old plant debris and on cucumber seed. During rains it splashes from the soil to the stems, leaves and later to the fruit. Once infection takes place, the organism is spread over the field on the hands of workers or by cucumber beetles. Angular leaf spot is most severe during extended rainy periods when temperatures are between 21 and 27°C.

CONTROL:

1. Every effort should be made to buy disease-free seed.
2. Never grow cucumbers on land where cucumbers, pumpkins or squash have been grown within the past three years.
3. Do not work in the crop when the foliage is wet.
4. Turn under crop refuse promptly after harvest.
5. Certain cucumber varieties are resistant to angular leaf spot. (See page 59).
6. Copper is recommended for control of angular leaf spot but has little effect on other cucumber diseases. Use formulations such as cupric hydroxide (Kocide 101 or Coppercide), tribasic copper sulphate (Tri-Cop) or copper oxychloride (C.O.C.S.). Preharvest interval = 1 day for all chemicals.

DAMPING-OFF

See RADISH - Damping Off

MOSAIC

PATHOGEN: Cucumber Mosaic Virus

SYMPTOMS: The first evidence of mosaic is a slight yellowing in the leaf veins. This is followed by a mottled pattern of irregularly shaped areas of dark green alternating with light green or yellow areas in the young expanding leaves. These symptoms are most evident on the tips of the runners. As the leaf matures, the mottling is less pronounced. Leaves that are mature or near maturity when infection occurs show little or no evidence of the disease. Cucumber mosaic virus also may cause mottling and distortion of the fruit and abortion of the flower buds. Wart-like areas are very pronounced in the slicer-type cucumbers. Although pickling varieties may be similarly affected, the infection often is characterized by fruit almost lacking in green color and referred to as "white pickle".

DISEASE CYCLE: Cucumber mosaic virus is spread by aphids, principally the green peach aphid. Aphids that feed on diseased plants either weed hosts or cucumbers, may acquire the virus and carry it to healthy plants, but soon lose the virus unless they feed again on infected plants.

CONTROL: Mosaic viruses are very difficult to control because they are widespread and transmitted in the field by common insect vectors. The use of resistant varieties is the only feasible means of control. (See page 59).

SCAB

PATHOGEN: *Cladosporium cucumerinum*

SYMPTOMS: Scab can be severe and epidemic in cool, foggy, wet seasons with cold nights. It appears on leaves in mid-season as small, circular to angular spots, water-soaked and pale brown. The disease is sometimes difficult to distinguish from angular leaf spot. However, scab lesions do not have a whitish crusty covering, and the veinlets remain brown against a paler background. In very humid conditions, the lesions have an olive-green covering of spores of the fungus in a velvety mat. These spores extrude through the stomata. Stems and young leaves become water-soaked and soon wilt and die back. At the vine tip, the disease sometimes resembles cucumber mosaic virus disease. However, the most serious damage occurs on the fruit. Small fruit have slightly sunken, water-soaked gray spots. These spots sometimes exude a gummy brown material which dries to a brown bead. As the fruit expand, the cavities enlarge often up to 1 cm in diameter and as much as 0.5 cm deep. Later, the fungus produces its spores in the velvety olive-green lining of the enlarged lesions. New lesions on the larger fruit become corked over and the shallow cavity is lined with a tan-colored scab.

DISEASE CYCLE: The *Cladosporium* fungus lives over winter in old cucumber refuse and can also be seed-borne. Spores are spread by wind, rain, insects and on clothing, tools, etc.

CONTROL:

1. Use disease-free seed, if possible.
2. Turn under crop refuse promptly after harvest.
3. Follow a three year rotation.
4. Some cucumber varieties are resistant to scab. (See page 59).
5. Apply protective sprays of one of the following fungicides: benomyl (Benlate), benomyl + mancozeb (Dithane M-45 or Manzate 200) or chlorothalonil (Bravo). Preharvest intervals - 1 day (benomyl, chlorothalonil), 5 days (benomyl + mancozeb).

WILT

PATHOGENS: *Fusarium* spp.

SYMPTOMS: Plants can be attacked in all stages of growth. When very young seedlings are invaded, they may damp-off and die or be stunted seriously and the cotyledon wilted. Older plants show a true wilting at first or, when somewhat resistant, may only be dwarfed. The wilting normally progresses slowly, showing only in the middle of the day. Later the edges of the leaves show tip burning until the entire plant dies. On the inside of the wilting stem, discoloration of the water ducts may show plainly or not at all. In wet weather the dead stems have a white or pinkish fungus growth on their surface. In advanced stages of the disease the roots begin to decompose and finally are killed.

DISEASE CYCLE: *Fusarium* lives from season to season in old diseased vines and is able to remain alive indefinitely in infested soil. The fungus enters the plant through the root tip or where side roots cause an opening. Injuries help the pathogen to penetrate the root system. After the mycellium enters the water ducts, the cavities become completely filled with the hyphae. The fungus sporulates after the vines are killed. There are three types of spores produced colorless globular spores (microconidia); large sickle-shaped spores (macroconidia); and thick-walled spores (chlamydospores). These spores and mycelium are disseminated with infested seed, soil and manure. They also are carried by flowing water.

CONTROL:

1. Use disease-free seed.
2. Start plants in steamed or fumigated soil mix.
3. Avoid fields known to be infested with Fusarium wilt.
4. Resistant varieties may be available from some seed companies.

MISCELLANEOUS DISORDERS
NUTRIENT DEFICIENCIES

Nitrogen - Plants grow poorly and usually are light green in color. Lower leaves turn yellow or light brown and the stems are short and slender.

Phosphorus - Young leaves are bluish-green with purple tints. The lower leaves sometimes turn light bronze with purple or brown spots. Shoots are short and thin, upright and spindly.

Potassium - Plants have thin shoots. Dieback may occur in severe cases. Older leaves may show slight chlorosis with typical browning of the tips, scorching of the margins and many brown spots, usually near the margins. Leaves may gradually roll upward and inward.

Magnesium - Older leaves are thin. The younger ones become mottled or chlorotic, followed by reddening and sometimes the appearance of necrotic spots. The tips and margins of leaves may turn downward and inward so that the leaves appear cupped. Defoliation may follow.

Calcium - Young leaves become distorted with the tips hooked back and the margins curled. New leaves may fail to unroll. Often the leaves are irregular in shape and ragged with brown scorching or spotting at the margins. Terminal buds eventually die if the deficiency is severe.

Sulphur - Plants have pale green or light yellow colored young leaves without spot formation. Symptoms may resemble those of nitrogen deficiency. The deficiency is common in areas with acidic, highly leached, sandy soils low in organic matter.

Boron - Bases of young leaves of terminal buds become light green and finally break down. Stems and leaves become distorted. Fruit may crack on the surface or rot in the center.

Zinc - Leaves may initially show interveinal chlorosis, then later develop necrotic areas and show purple pigmentation. Leaves are few and small, with short internodes. Poor fruit production is a common symptom of zinc deficiency.

Copper - Younger leaves become pale green with necrotic areas between the veins.

Manganese - Leaf symptoms are similar to copper deficiency, except that small leaves are apt to become crinkled. This deficiency is most common on high pH soils.

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IRON DEFICIENCY IN PLANTS

GENERAL

Under certain soil conditions, iron deficiency may occur in fruit trees, ornamentals and other plants. Although iron is the most abundant of the trace elements, the availability of iron in the soil may be low. As yet there is no suitable soil test for iron, as there is for the major nutrients. Diagnosis of iron deficiency, therefore, rests on identifiable plant symptoms plus a knowledge of the history of the soil.

FACTORS ENCOURAGING IRON DEFICIENCY

- Excess lime in the soil, "lime-induced chlorosis"
- High soil pH (above 8.0)
- Bicarbonate ions in the soil or irrigation water
- Excess amount of phosphates in the soil
- Excess soil moisture and low soil temperatures
- Excess quantities of metallic ions such as copper and manganese which may occur in acid soils

SYMPTOMS

Iron deficiency is characterized by chlorosis (yellowing) of the younger leaves of many species. The tissue between the veins gradually turns yellow while the veins tend to stay green. The conditions may advance throughout the entire plant if unchecked and the tips and margins of some leaves may turn brown and become dry and brittle. In severe cases and with certain species, the new leaves may lose all their green color and appear straw-colored.

Plant species vary in their susceptibility to iron deficiency:

Susceptible	Relatively Tolerant
Raspberry	Russian Olive
Mountain Ash	Lilac
Spiraea	Cotoneaster
Apple	Spruce
Currant	Barberry
High Bush Cranberry	
Elder	

IRON DEFICIENT LEAVES

Network of veins usually retain their green color while areas between veins become chlorotic.

Conifers such as pine, fir and juniper are less susceptible but may show general needle yellowing on the upper or youngest clusters. Iron deficiencies rarely occur in forage grasses or legumes. Cereal crops may show an intervenal chlorosis or striping along the leaf. Lawn grasses may occasionally show iron deficiency because of high pH or high levels of lime or phosphate in the soil.

AREAS AFFECTED IN ALBERTA

Although iron deficiency may occur in localized areas throughout the province depending on the factors noted above, the condition is most likely to show up in susceptible species growing on high lime or calcareous soils. These soils are found mainly in southern Alberta, primarily in the Brown Soil zone. The closer the lime layer to the surface, as in the case of eroded soils, the greater is the likelihood of induced iron deficiency. In southwestern Alberta there is a wide band of high lime soils extending south of Calgary through the Claresholm area to Fort Macleod.

DIAGNOSIS

1. The application of a foliar spray of an iron-containing compound may be the best means of verifying the diagnosis (see below).

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2. Soil tests specifically for iron are not recommended, but they will determine factors such as pH and lime content which might be affecting the availability of iron to plants.
3. Plant analysis for iron content is of limited value. Soil dust on the sample may be a serious source of iron contamination. Also, plants showing iron chlorosis induced by high levels of phosphorus generally have a normal concentration of iron in the tissues. Such iron, however, does not appear to be readily available to the plants.

TREATMENT

Soil treatments for iron deficiency are not always successful since the same factors that limit the availability of soil iron also render added iron unavailable to plants. The use of foliar sprays and chelated iron compounds, however do offer some promise. When deficiencies exist, iron-chlorotic leaves should begin to "green-up" within ten days after application of iron.

In the case of greenhouse crops particularly, proper adjustment of the pH of the growing medium is often the best preventive treatment. For example, azaleas are grown in a medium-high acid peat which helps to ensure the availability of iron to meet the high iron requirement of these plants.

Soil Application of iron salts, particularly on calcareous soils, is not recommended unless chelated products are available. The chelate resists efforts in the soil to render its iron unavailable to plants.

For trees and shrubs, soil application is most effective if the chelate is applied to the soil and watered in around the base. For potted plants the iron may be dissolved in water and applied as a soil drench.

Iron compounds may be purchased from garden supply centres. A range of package sizes is available. The iron may be in chelate or micronized form and varies in concentration from product to product. As a result of this, general application rates cannot be given here. Purchasers should follow the information supplied on the product label.

Foliar Sprays of iron salts have been used successfully. A 1–2% solution of ferrous sulfate (0.5–1 kg per 45 L of water) may be applied to iron deficient plants. One litre per 20 square metres, or on a field scale, 170–225 L per acre should be adequate. The addition of a wetting agent: i.e. several drops of a mild detergent per gallon is essential.

Iron chelates can also be used in foliar sprays instead of soil applications.

CONCLUSIONS

It should be noted that foliar applications of any iron compound bring about temporary correction of iron chlorosis in a plant, but only by correcting the soil conditions responsible can permanent recovery ever be obtained.

The most practical solution to "lime-induced" chlorosis is learning to live with it and to recognize that a causative factor that induces lime chlorosis may not easily be removed once it is established in a soil. This means that steps should be taken to alleviate, not aggravate, the condition and plant species should be selected which are resistant to iron chlorosis.

Metric Conversion

1 pound X 0.45 = 1 kg
1 gallon X 4.5 = 1 litre
1 square foot X 0.09 = 1 square metre

2,4-D INJURY

SYMPTOMS: Many plants, epsecially tomatoes, are susceptible to 2,4-D injury. The first symptom is a downward curvature of the leaves and the tips of the growing points. Even minute amounts of 2,4-D drift may cause rather noticeable amounts of distortion of growth. In more severe cases stems become thick, stiff and brittle and show a whitening of the external surface accompanied by a severe curvature and twisting of the leaves. New leaves do not expand fully and are elongated with abnormally pointed tips. The veins become prominent, light green and appear parallel. Portions of the tissues between the veins often appear as dark green spots. In tomato fruits, inner parts remain green while externally the fruits appear to be ripe. Ripe fruits are harder than usual to remove from the affected plants.

CONTROL:

1. Do not spray when the wind may carry spray drift toward tomatoes or other sensitive crops.

2. Use the amine form of 2,4-D and apply the spray at a low pressure witha coarse nozzle to minimize drift.

3. Because it is almost impossible to remove 2,4-D from a sprayer by washing and rinsing after use, use a separate sprayer to apply insecticides and fungicides.

GROWTH CRACKING

CAUSE: A rapid swelling or growth generally caused by sudden changes in soil moisture. It is most common after heavy rains that follow a dry spell. Improper timing of irrigation may also cause growth cracking.

SYMPTOMS: Longitudinal cracks may extend from the crown to lower portions of the roots in the cases of root crops. Heads of cabbage may split open across the top. Superficial or deep cracks allow tissue or storage organs to dry out. Occasionally these wounds may callus over. Soft rot bacteria and soil fungi frequently enter the cracks and cause a decay.

CONTROL:

1. Regulate soil moisture to prevent dry-wet cycles especially as crops near maturity.
2. Some vegetables, e.g., cabbage, may be lifted slightly to tear off feeder roots therby reducing the plant's ability to rapidly take up large quantities of water.
3. Avoid rough handling of crops during harvest.

DISEASE-RESISTANT VEGETABLES

Vegetable	Variety	Disease Reaction
Asparagus	Brock's Imperial Hybrid	R
	Mary Washington	R
	Mary Washington Improved	R
	Paradise	R
	Viking 2K	F
	Viking	R

Disease resistance or tolerance: R = Rust; F = Fusarium

Beans	Blue Lake Bush 47	CBM, V
	Blue Lake Bush 274	CBM, V
	Blue Lake Stringless	CBM
	Contender	CBM, PM
	Dade	CBM, R
	Del Ray	CBM
	Executive	CBM
	Goldcrop (wax)	CBM, V
	Provider	CBM, PM
	Golden Wax Improved	CBM
	Gold Rush	CBM, V
	Greencrop	CBM, PM
	Harvester	CBM, V
	Honeygold	CBM
	Improved Tendergrass	CBM
	Midas	CBM, V
	Sungold (wax)	CBM, V
	Tendercrop	CBM, PM, V
	Topcrop	CBM, V

Disease resistance or tolerance: CBM = common bean mosaic; PM = powdery mildew; R = rust; V = other viruses.

Broccoli	Futura	DM
	Green Comet	H
	Premium Crop	H

Disease resistance or tolerance: DM = downy mildew; H = heat tolerant

Cabbage	Blueboy	F, BR
	Blue Heaven	BS, F
	Bravo	F, BR
	Canada Kraut	F
	Enterprise	F
	Express	F
	Golden Acre	F
	Gourmet	F
	Hybrid Jet Pak	F
	King Cole	F
	Market Prize	F
	Market Topper	F
	Market Victor	F

Cabbage cont'd	Princess	F
	Redhead	F
	Roundup	F
	Safekeeper	BR
	Stokes Viking Golden Acre	F
	Stonehead Hybrid	F
	Sun-Up	F
	Supermarket	BR, BS, F
	Tastie	F

Disease resistance or tolerance: BR = black rot; BS = black speck; F = Fusarium yellows.

Cantaloupe (Muskmelon)		
	Alaska	F
	Canada Gem Hybrid	F, PM
	Delicious 51	F, PM
	Honey Rock	F
	Iroquois	F
	Mainerock Hybrid	F

Disease resistance or tolerance: PM = powdery mildew; F = Fusarium wilt.

Carrot	Chancellor	RR, A
	Fancipak	B
	Paramount	A
	Sierra	B
	Spartan Premium	RR

Disease resistance or tolerance: A = Alternaria; B = bolt; RR = rusty root.

Cucumber	Bounty	ALS, An, DM,M,PM,S
	Dasher	ALS,An,DM, M, PM, S
	Explorer	ALS,DM,PM
	High Mark II	DM,M,S
	Marketmore 70	M, S
	Peppi	DM,PM,S
	Pioneer Hybrid	ALS,An,DM, PM,S
	Slicemaster F-1	ALS,An,DM, M,PM,S
	Spartan Dawn	M,S
	Sweet Slice Hybrid	ALS,An,DM, M,PM,S
	Victory	ALS,An,DM, M,PM,S

Disease resistance or tolerance: ALS = angular leaf spot; An = Anthracnose; DM = downy mildew; PM = powdery mildew; M = mosaic; S = scab.

Onion	Stokes Explorer	F

Disease resistance or tolerance; F = Fusarium

Pea	Sugar Snap	F
	Green Arrow	DM, F, V
	Novella	F
	Progress No. 9	F
	Thomas Laxton	F
	Venus	F

Disease resistance or tolerance; DM = downy mildew; F = Fusarium wilt; V = virus.

Green Peppers	Big Bertha	TMV
	California Wonder	TMV
	Early Niagra	TMV
	Gypsy	TMV
	Mercury	TMV
	Staddon's Select	TMV
	Yolo Wonder	TMV

Disease resistance or tolerance; TMV = tobacco mosaic virus.

Potato	Chieftain	S
	Norchip	S
	Norgold Russet	S
	Norland	S
	Russet Burbenk (Netted Gem)	S
	Superior	S
	Viking	S

Disease resistance or tolerance; S = scab

Sweet Corn (bicolor)	Butter and Sugar	BW, SCLB
	Calico	BW, SCLB
Sweet Corn (white)	Platinum Lady	BW
	Silver Queen	BW
	White Lightning	MDM

Disease resistance or tolerance; BW = bacterial wilt; MDM = maize dwarf mosaic; SCLB = southern corn leaf blight; S = smut.

Tomato	Earlirouge	V
	Basket Vee	V
	Hybrid 31	ASC, F, V
	Nova	F
	Peto 95	ASC, F, V
	Roma	F, V
	The Juice	F, V
	Ultra Girl	F, N, V

Disease resistance or tolerance; ASC = Alernaria stem canker; F = Fusarium race 1; N = root-knot nematode; V = Verticillium wilt.

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Canada Canada
metric métrique

METRIC CONVERSION TABLE

Approximate conversions to metric measures

When You Know	Multiply by	To Find	Symbol
LENGTH			
inches	2.5	centimetres	cm
feet	0.3	metres	m
yards	0.9	metres	m
miles	1.6	kilometres	km
AREA			
square inches	6.5	square centimetres	cm^2
square feet	0.09	square metres	m^2
square yards	0.8	square metres	m^2
square miles	2.6	square kilometres	km^2
acres	0.4	hectares	ha
MASS			
ounces	28	grams	g
pounds	0.45	kilograms	kg
short tons (2000 lb)	0.9	tonnes	t
VOLUME			
fluid ounces	28	millilitres	ml
pints	0.57	litres	l
quarts	1.14	litres	l
gallons	4.5	litres	l
cubic feet	0.03	cubic metres	m^3
cubic yards	0.76	cubic metres	m^3
TEMPERATURE (exact)			
Fahrenheit		Celsius	°C

°F - 32 x 5/9 = °C

TABLE DE CONVERSION MÉTRIQUE

Conversion approximative aux unités métriques de mesure

Lorsque vous connaissez les mesures en	Multipliez par	Pour obtenir	Symbole
LONGUEUR			
pouces	2.5	centimètres	cm
pieds	0.3	mètres	m
verges	0.9	mètres	m
milles	1.6	kilomètres	km
SUPERFICIE			
pouces carrés	6.5	centimètres carrés	cm^2
pieds carrés	0.09	mètres carrés	m^2
verges carrées	0.8	mètres carrés	m^2
milles carrés	2.6	kilomètres carrés	km^2
acres	0.4	hectares	ha
MASSE			
onces	28	grammes	g
livres	0.45	kilogrammes	kg
tonnes courtes (2000 lb)	0.9	tonnes	t
VOLUME			
onces liquides	28	millilitres	ml
chopines	0.57	litres	l
pintes	1.14	litres	l
gallons	4.5	litres	l
pieds cubes	0.03	mètres cubes	m^3
verges cubes	0.76	mètres cubes	m^3
TEMPÉRATURE (exacte)			
Fahrenheit		Celsius	°C

°F - 32 x 5/9 = °C

Metric Commission

Commission du système métrique

Simple Conversion Table of Length

Inches	1	2	3	4	5	6	7	8	9	10	11	12	
Centimetres	2.54	5.08	7.62	10.16	12.70	15.24	17.78	20.32	22.86	25.40	27.94	30.48 (approx 0.3 m)	
Feet	1	2	3	4	5	6	7	8	9	10			
Metres	0.3	0.6	0.9	1.2	1.5	1.8	2.1	2.4	2.7	3.0			
Miles	1	2	3	4	5	6	7	8	9	10			
Kilometres	1.6	3.2	4.8	6.4	8.0	9.7	11.3	12.9	14.5	16.1			

Simple Conversion Table of Weight

Ounces (av)	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
Grams	28.3	56.7	85.0	113.4	141.7	170.1	198.4	226.8	255.1	283.5	311.8	340.2	368.5	396.9	425.2	453.6
Pounds	1	2	3	4	5	6	7	8	9	10						
Kilograms	0.45	0.91	1.36	1.81	2.27	2.72	3.18	3.63	4.08	4.54						

(approx 0.45 kg)

Simple Conversion Table of Temperature

Degrees Fahrenheit	0	10	20	30	32°	40	50	60	70	80	90	100	200	212	400
Degrees Celsius	−17.8	−12.2	−6.7	−1.1	0°	4.4	10.0	15.6	21.1†	26.7	32.2	37.8	93.3	100.0	204.4‡

*(Water Freezes)
†(Room Temperature)
‡(Hot Oven)

Simple Conversion Table of Area

Square Inches	1	2	3	4	5	6	7	8	9	10
Square Centimetres	6.45	12.90	19.36	25.81	32.26	38.71	45.16	51.61	58.06	64.52
Square Feet	1	2	3	4	5	6	7	8	9	10
Square Metres	0.09	0.19	0.28	0.37	0.46	0.56	0.65	0.74	0.84	0.93
Acres	1	2	3	4	5	6	7	8	9	10
Square Metres	4 047	8 094	12 141	16 187	20 234	24 281	28 328	32 375	36 422	40 469

Simple Conversion Table of Volume

Ounces (fl.)	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
Millilitres	28.4	56.8	85.2	113.7	142.1	170.5	198.9	227.3	255.7	284.1	312.5	341.0	369.4	397.8	426.2	454.6
Ounces (fl.)	17	18	19	20												
Millilitres	483.0	511.4	539.8	568.3 (approx. 0.57 *l*)												
Pints	1	2	3	4	5	6	7	8	9	10						
Litres	0.57	1.14	1.70	2.27	2.84	3.41	3.98	4.55	5.11	5.68						
Gallons	1	2	3	4	5	6	7	8	9	10						
Litres	4.5	9.1	13.6	18.2	22.7	27.3	31.8	36.4	40.9	45.5						

POOR

QUALITY

ORIGINAL